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L5 ANSWER 1 OF 7 MEDLINE on STN

TI Gastric secretion.
AB Overlapping neural, hormonal, and paracrine pathways finely regulate gastric acid secretion. In rats and guinea pigs, most of the intrinsic neural innervation to the gastric mucosa originates in the myenteric plexus. In contrast, human stomachs have a clearly defined submucosal plexus that contains a variety of transmitters including nitric oxide, vasoactive intestinal peptide (VIP), gastrin-releasing peptide (GRP), substance P, and calcitonin gene-related peptide (CGRP). Although GRP is known to participate in meal-stimulated acid secretion by releasing gastrin in a variety of laboratory animals, recent studies were unable to demonstrate a role for endogenous GRP in meal-stimulated gastrin secretion in humans. Pituitary adenylate cyclase-activating polypeptide (PACAP), a member of the secretin-glucagon-VIP family, has been localized to gastric mucosal neurons and may participate in vagally mediated acid secretion. Two novel peptides, ghrelin and leptin, have been localized to the stomach. Peripheral administration of ghrelin stimulates and of leptin inhibits acid secretion. The binding of secretagogues to parietal cells generates changes in second messengers that regulate the translocation and activation of the proton pump, H_K-ATPase. In resting cells, H_K-ATPase is contained within cytoplasmic tubulovesicles in an inactive form. At stimulation, the tubulovesicles fuse with the apical canalicular and the H_K-ATPase is incorporated into the apical membrane where it actively pumps H ions in exchange for K. Acute infection with Helicobacter pylori results in hypochlorhydria, whereas chronic infection can cause either hypo- or hyperchlorhydria, depending on the distribution of the infection and the degree of corpus gastritis. Recent studies suggest that inflammatory cytokines, produced in response to the organism, can play a role in the perturbations in acid and gastrin secretion induced by H. pylori.
SO Current opinion in gastroenterology, (2002 Nov) Vol. 18, No. 6, pp. 639-49.
Journal code: 8506887. ISSN: 0267-1379.

L5 ANSWER 2 OF 7 MEDLINE on STN
TI Is microvascular flow rate related to ghrelin, leptin and adiponectin levels?.
AB Ghrelin, leptin and adiponectin are three hormones which are frequently associated with metabolism, obesity and appetite. Recently, it has been shown that they may possess other physiologic roles, specially in connection with the circulation. Ghrelin infusion increases forearm blood-flow in a dose-dependent manner. Leptin has been shown to be involved not only in thermogenesis but angiogenesis as well. Adiponectin, apart from its insulin-sensitizing action, appears to modulate inflammation by inhibiting monocyte adhesion to endothelial cells. Six monkeys, which had been classified as being in the pre-diabetic state, where administered a triglyceride lowering regimen. Microvascular function was assessed using a laser Doppler flow-meter during a temperature provocation test. Percent change in flow from baseline following temperature elevation, as well as percent change in flow/degree rise in temperature were used to evaluate microvascular reserve and reactivity. Using univariate analysis, it appears that increased perfusion is significantly correlated with adiponectin, followed by leptin. Flow was also positively correlated with ghrelin, but the relationship did not attain significance. As expected, flow was also negatively and significantly correlated with fibrinogen. Trends show that flow was also negatively correlated to circulating triglyceride levels ($p=0.08$). The data indicate that the three hormones appear to possess microvascular actions that may impact on their other physiologic functions.
SO Clinical hemorheology and microcirculation, (2003) Vol. 29, No. 3-4, pp. 409-16.
Journal code: 9709206. ISSN: 1386-0291.

L5 ANSWER 3 OF 7 MEDLINE on STN
TI Ghrelin attenuates the development of acute pancreatitis in rat.
AB BACKGROUND: Ghrelin, a circulating growth hormone-releasing peptide isolated from human and rat stomach, stimulates growth hormone secretion, food intake and exhibits gastroprotective properties. Ghrelin is predominantly produced by a population of endocrine cells in the gastric mucosa, but its presence in bowel, pancreas, pituitary and hypothalamus has been reported. In human fetal pancreas, ghrelin is expressed in a prominent endocrine cell population. In adult pancreatic islets the population of these cell is reduced. The aim of present study was to investigate the influence of ghrelin administration on the development of acute pancreatitis. METHODS: Acute pancreatitis was induced in rat by caerulein injection. Ghrelin was administrated twice (30 min prior to the first caerulein or saline injection and 3 h later) at the doses: 2, 10 or 20 nmol/kg. Immediately after cessation of caerulein or saline injections the following parameters were measured: pancreatic blood flow, plasma lipase activity, plasma interleukin-1beta (IL-1beta) and interleukin 10 (IL-10) concentration, pancreatic DNA synthesis, and morphological signs of pancreatitis. RESULTS: Administration of ghrelin without induction of pancreatitis did not affect significantly any parameter tested. Caeerulein led to the development of acute edematous pancreatitis. Treatment with ghrelin at the dose 2 nmol/kg, during induction of pancreatitis, was without effect on pancreatic histology or biochemical and functional parameters. Treatment with ghrelin at the dose 10 and 20 nmol/kg attenuated the development of pancreatitis and the effects of both doses were similar. Administration of ghrelin (10 or 20 nmol/kg) reduced inflammatory infiltration of pancreatic tissue and vacuolization of acinar cells. Also, plasma lipase activity and plasma IL-1beta concentration were reduced, and caerulein-induced fall in pancreatic DNA synthesis was reversed. Administration of ghrelin at the dose 10 and 20 nmol/kg was without effect on caerulein-induced pancreatic edema and pancreatitis-related fall in pancreatic blood flow. CONCLUSIONS: (1) Administration of ghrelin attenuates pancreatic damage in caerulein-induced pancreatitis; (2) Protective effect of ghrelin administration seems Background: Ghrelin, a circulating growth hormone-releasing peptide isolated from human and rat stomach, stimulates growth hormone secretion, food intake and exhibits gastroprotective properties. Ghrelin is predominantly produced by a population of endocrine cells in the gastric mucosa, but its presence in bowel, pancreas, pituitary and hypothalamus has been reported. In human fetal pancreas, ghrelin is expressed in a prominent endocrine cell population. In adult pancreatic islets the population of these cell is reduced. The aim of present study was to investigate the influence of ghrelin administration on the development of acute pancreatitis. Methods: Acute pancreatitis was induced in rat by caerulein injection. Ghrelin was administrated twice (30 min prior to the first caerulein or saline injection and 3 h later) at the doses: 2, 10 or 20 nmol/kg. Immediately after cessation of caerulein or saline injections the following parameters were measured: pancreatic blood flow, plasma lipase activity, plasma interleukin-1beta (IL-1beta) and interleukin 10 (IL-10) concentration, pancreatic DNA synthesis, and morphological signs of pancreatitis. Results: Administration of ghrelin without induction of pancreatitis did not affect significantly any parameter tested. Caeerulein led to the development of acute edematous pancreatitis. Treatment with ghrelin at the dose 2 nmol/kg, during induction of pancreatitis, was without effect on pancreatic histology or biochemical and functional parameters. Treatment with ghrelin at the dose 10 and 20 nmol/kg attenuated the development of pancreatitis and the effects of both doses were similar. Administration of ghrelin (10 or 20 nmol/kg) reduced inflammatory infiltration of

pancreatic tissue and vacuolization of acinar cells. Also, plasma lipase activity and plasma IL-1beta conc; concentration were reduced, and caerulein-induced fall in pancreatic DNA synthesis was reversed. Administration of ghrelin at the dose 10 and 20 nmol/kg was without effect on caerulein-induced pancreatic edema and pancreatitis-related fall in pancreatic blood flow. Conclusions: (1) Administration of ghrelin attenuates pancreatic damage in caerulein-induced pancreatitis; (2) Protective effect of ghrelin administration seems to be related the inhibition in inflammatory process and the reduction in liberation of pro-inflammatory IL-1beta.

SO Journal of physiology and pharmacology : an official journal of the Polish Physiological Society, (2003 Dec) Vol. 54, No. 4, pp. 561-73.
Journal code: 9114501. ISSN: 0867-5910.

L5 ANSWER 4 OF 7 MEDLINE on STN
TI Is obesity an inflammatory condition?.
AB Obesity may be a low-grade systemic inflammatory disease. Overweight and obese children and adults have elevated serum levels of C-reactive protein, interleukin-6, tumor necrosis factor-alpha, and leptin, which are known markers of inflammation and closely associated with cardiovascular risk factors and cardiovascular and non-cardiovascular causes of death. This may explain the increased risk of diabetes, heart disease, and many other chronic diseases in the obese. The complex interaction between several neurotransmitters such as dopamine, serotonin, neuropeptide Y, leptin, acetylcholine, melanin-concentrating hormone, ghrelin, nitric oxide, and cytokines and insulin and insulin receptors in the brain ultimately determines and regulates food intake. Breast-feeding of more than 12 mo is associated with decreased incidence of obesity. Breast milk is a rich source of long-chain polyunsaturated fatty acids (LCPUFAs) and brain is especially rich in these fatty acids. LCPUFAs inhibit the production of proinflammatory cytokines and enhance the number of insulin receptors in various tissues and the actions of insulin and several neurotransmitters. LCPUFAs may enhance the production of bone morphogenetic proteins, which participate in neurogenesis, so these fatty acids might play an important role in brain development and function. It is proposed that obesity is a result of inadequate breast feeding, which results in marginal deficiency of LCPUFAs during the critical stages of brain development. This results in an imbalance in the structure, function, and feedback loops among various neurotransmitters and their receptors, which ultimately leads to a decrease in the number of dopamine and insulin receptors in the brain. Hence, promoting prolonged breast feeding may decrease the prevalence of obesity. Exercise enhances parasympathetic tone, promotes antiinflammation, and augments brain acetylcholine and dopamine levels, events that suppress appetite. Acetylcholine and insulin inhibit the production of proinflammatory cytokines and provide a negative feedback loop for postprandial inhibition of food intake, in part, by regulating leptin action. Statins, peroxisome proliferator-activated receptor-gamma binding agents, non-steroidal antiinflammatory drugs, and infant formulas supplemented with LCPUFAs, and LCPUFAs themselves, which suppress inflammation, may be beneficial in obesity.

SO Nutrition (Burbank, Los Angeles County, Calif.), (2001 Nov-Dec) Vol. 17, No. 11-12, pp. 953-66. Ref: 230
Journal code: 8802712. ISSN: 0899-9007.

L5 ANSWER 5 OF 7 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN
TI Effect of lipopolysaccharide administration of peripheral ghrelin levels in rats.
AB Background: Infection and inflammation have been known to decrease appetite and cause significant weight loss in humans. Hormones

that regulate food intake may be implicated in this process. Ghrelin, a peptide growth hormone secretagogue produced mainly in the stomach, has been recently shown to stimulate feeding with increased secretion. Aims: In order to study the changes in appetite and ghrelin secretion under inflammation, we measured food intake and plasma ghrelin levels in fasted rats after the peripheral administration of lipopolysaccharide (LPS), an endotoxin which is a component of the outer membrane of Gram-negative bacteria, that induces an inflammatory response. Methods: LPS was injected intraperitoneally (ip) at 100 (g/kg in overnight-fasted Sprague-Dawley rats. The blood samples were taken before and 3 h after injection and in a subset of rats food intake was observed 2 h after injection for 1h. Ghrelin levels were measured in plasma by radioimmunoassay. Results: Our results show that LPS injected ip reduced food intake as compared to rats injected with vehicle ip alone (1.74(0.51 vs. 4.88(1.63 g/h). Ghrelin secretion into the circulation was also depressed by 90% (4% three hours after LPS administration. Conclusions: These results demonstrate that inhibition of ghrelin secretion may be one mechanism by which anorexia is produced in patients suffering from infectious processes. This study was supported by NIDDK Grant 3 T32 DK07688.

SO FASEB Journal, (March 2003) Vol. 17, No. 4-5, pp. Abstract No. 523.4. <http://www.fasebj.org/>. e-file.

Meeting Info.: FASEB Meeting on Experimental Biology: Translating the Genome. San Diego, CA, USA. April 11-15, 2003. FASEB.
ISSN: 0892-6638 (ISSN print).

L5 ANSWER 6 OF 7 CAPLUS COPYRIGHT 2008 ACS on STN
TI Use of GLP-2 or its analogs for the treatment or prevention of bone-related disorders
AB The present invention relates to methods for prevention and treatment of bone-related using a GLP-2 mol. or GLP-2 activator either alone or in combination with another therapeutic. The invention also encompasses methods of monitoring the effectiveness of treatment of the invention.
SO U.S. Pat. Appl. Publ., 50pp., Cont.-in-part of U.S. Ser. No. 35,826.
CODEN: USXXCO

L5 ANSWER 7 OF 7 CAPLUS COPYRIGHT 2008 ACS on STN
TI Diagnosis and treatment of human dormancy-related sequellae
AB New methods for diagnosis and treatment of human dormancy syndrome-related sequellae are provided. Human dormancy syndrome (HDS) is characterized by elevated serum ratio of rT3/fT3 compared to a population of normal subjects. HDS includes fibromyalgia, chronic fatigue, cancer, autoimmune disease, obesity and related dormancy conditions. Dormancy and HDS-related sequellae are imposed on humans by infection with lipopolysaccharide (LPS; or endotoxin)-producing organisms, especially those that are intracellular and those that create antigens that stimulate the TLR pathways. In such instances, the elimination or neutralization of the LPS signal along with the infectious source is required to impact the sequellae of HDS. Treatment includes use of novel and non-obvious doses of antibiotics, optionally including agents that decrease the adverse effects of endotoxin.
SO U.S. Pat. Appl. Publ., 35 pp., Cont.-in-part of U. S. Ser. No. 444,845.
CODEN: USXXCO

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L6 0 CHARALABOS?/AU AND POTHOUAKIS?/AU
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